enantiomorphous shapes. In the case of infinite series the possible solutions may not be enantiomorphous. An example of two different pyramidal bodies is considered. Another type of ambiguity is associated with numerical instability of solution. The dependence of solution dispersion from the maximum scattering angle is considered. Several numerical examples which demonstrate an optimum data angular range existence are given. The main conclusion that prior to experimental data interpretation one should to perform modelling of the expected structure to estimate the degree of ambiguity and to choose both data weighting functions and angular range is shown by the results of model calculations.

Keywords: *ab-initio* structure determination, nonlinear optimization, numerical methods and simulation techniques

P03.10.30

Acta Cryst. (2008). A**64**, C227

Development of a scoring method for predicting protein complex structures

Yuko Tsuchiya[1], Eiji Kanamori[2], Daron M Standley[3], Haruki Nakamura[3], Kengo Kinoshita[1]

[1]Institute of Medical Science, the University of Tokyo, Human Genome center, 4-6-1 Shirokane-dai, Minato-ku, Tokyo, 108-8639, Japan, [2]Biomedicinal Information Research Center, 2-41-6 Aomi, Koto-ku, Tokyo, 135-0064, Japan, [3]Institute for Protein Research, Osaka University, 3-2 Yamadaoka, Suita, Osaka, 565-0871, Japan, E-mail：yukoo@hgc.jp

The information about protein-protein interactions increases much more rapidly than the increase of the number of the tertiary structures of those protein complexes. Therefore, precise prediction of protein complex structures by protein-protein docking simulations is required. When the protein complex is re-built from its component protomers which derive from experimentally determined complex structure (native structure) by docking, the complex models with rmsd < 10Å from the native structure (near-native model) could be obtained, along with a great number of false positives (decoy). The separation of near-native models from many decoys is therefore needed in the prediction of complex structures by docking. In this study, we developed the method for scoring docking models so that the near-native models were higher in rank than decoys, based on the assumption that the interfaces of near-native models are more complementary in terms of surface properties and shapes compared to those of decoys. We used 125 non-redundant hetero-dimers (native structures) as targets. For each target, maximum 500 complex models were generated by our docking method. We also observed these targets in terms of the shape of the interfaces of their native structures. As a result, we found that these targets could be classified into two groups according to their interface shapes, and moreover, that this classification correlated with another classification which was based on the number of models with high docking score, namely, the difficulty in the separation of near-native models. We therefore only focused on 75 targets classified as difficult targets which need the separation. So far our method could separate the near-native models from the decoys in 70% of these targets.

Keywords: complex structure prediction, protein-protein docking, analysis of protein-protein interfaces

P03.10.31

Acta Cryst. (2008). A**64**, C227

3D homology structure model for a pyrazinamide susceptibility test in *Mycobacterium tuberculosis*

Luis R Castillo

Universidad Peruana Cayetano Heredia, Biochemistry, Molecular Biology and Pharmacology/Unidad de Bioinformatica, Av. Honorio Delgado 430, Urb. Ingeniería, San Martin de Porres, Lima, Lima 31, Peru, E-mail：eloluis87@yahoo.com

Pyrazinamide (PZA) constitutes one of the First-Line Drugs for Tuberculosis treatment and appears to be the most important drug killing the latent *M. tuberculosis*. It is hydrolyzed by *Mycobacterium tuberculosis* pyrazinamidase / nicotinamidase (PZAse) to pyrazinoic acid (POA), the bactericidal agent. X-Ray fluoresecence Spectroscopy analysis shows that PZAse is a metalloenzyme that contains Zn^{+2}, and metal depletion of PZAse using EDTA decreases significantly its activity. Analysis of all PZA resistant strains reported in Peru and Worldwide shows that 90% of resistance are due to single point missense mutations on the PZAse sequence which are not homogeneously distributed along PZAse sequence, but clustered in the region near to the hypothetic PZAse Metal Coordination Site (MCS). Further analysis evaluating distinct amino acid physicochemical parameters and relative distances among the mutated residues in the PZAse 3D Homology Model shows that, those strains which have the highest PZA resistance(>1200 Minimum Inhibitory Concentration (MIC)), have mutations on residues which side chains aim at the MCS ($p<0.02$). These data indicate that PZAse coordinates, at least, a metal ion (Zn^{+2}), which is required for its enzymatic function, and the presented PZA Resistance Prediction Model, which is based on the analysis of the PZAse MCS, could be used in Low-Income countries for rapid and low-cost PZA susceptibility tests.

Keywords: pyrazinamidase (PZA), *Mycobacterium tuberculosis*, homology modelling of proteins

P03.10.32

Acta Cryst. (2008). A**64**, C227-228

Homology modeling of *Arabidopsis thaliana* glycolipid transfer protein

Lenita Viitanen, Tiina A Salminen

Abo Akademi University, Biochemistry, Tykistokatu 6, Turku, Turku, 20520, Finland, E-mail：lenita.viitanen@abo.fi

Glycolipid transfer protein (GLTP) is a 24 kDa cytosolic protein that can transfer glycolipids between membranes *in vitro*, but its function *in vivo* is still unknown. GLTPs have been found in various organisms and recently also in plants. Three different forms of GLTP are expressed in *Arabidopsis thaliana*: AtGLTP_1, AtGLTP_2 and AtGLTP_3. Our collaborators have tested the lipid transfer preferences of the AtGLTPs with a transfer assay. Using the known crystal structures of human GLTP [1, 2] as templates, I have constructed homology models of the AtGLTPs in complex with glycolipid ligands. I have studied and compared the models and templates in order to find structural characteristics that explain the differences in lipid transfer preference of GLTPs. The project is done in collaboration with the research groups of Dr. Johan Edqvist (Linköping University) and Dr. Peter Mattjus (Åbo Akademi University).

[1] Malinina et al. (2004) Nature, 430, 1048-1053
[2] Malinina et al. (2006) PLoS Biol, 11, e362